The sheet will probably be of the greatest usefulness in the so-called out-patient "classes," at which its meaning and use can be explained to groups of patients. One change might be made in the direction of greater simplicity. It will be seen that the size of the portions of fish and meats is a little different for each kind, but all are close to two cubic inches. It would perhaps be better to have a uniform portion for all meat and fish, that is, the lean part of a lamb chop or a piece 2″ x 1″ x 1″. This is an amount easily visualized, and a block of wood could even be cut in the requisite dimensions and shown as an object lesson.

With the varied diet which the patient will naturally choose for himself under this system he will usually get sufficient calories for his needs. That this is so will be discovered if he is told at intervals to bring in a list of the foods he has eaten on two or three days previous. When, however, his protein has been cut down to the neighborhood of 30 grams a day it may well be found that he is not getting calories enough. If so a definite daily amount of butter, cream, olive oil and sugar can be prescribed, with instructions to take them on his other food. For convenience of estimation, on the physician's own sheet the calories may be written in after each portion.

Finally, this scheme, as will be readily apparent, is based essentially on the diet chart published years ago by Arnold, which in the opinion of the writers should have met with a more general use than so far as they know, has been the case.[1] It was Arnold's great merit to point out that uniform amounts of protein can be taken by common measures in quantities of food which are about what one would naturally take as a serving. Arnold's charts, however, as we think anyone who has used them will agree, are of more service to the physician than to the patient. The scheme submitted herewith makes the data available as a guide for the patient and the physician.

We gratefully acknowledge the assistance of Miss Katharine Mitchell, formerly assistant dietitian at the Peter Bent Brigham Hospital.

ANTIPNEUMOCOCCUS SERUM (KYES'S) IN THE TREATMENT OF PNEUMONIA.[1]

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In 1911, Kyes[2] announced that he had succeeded in producing an antipneumococcus serum with high antibody content by a new method. The novelty lay in using an insusceptible host, the ordinary barnyard fowl, which circumstance allowed the use of unusually large numbers of virulent living pneumococci as antigen. It was, of course, a well-known fact in immunology that pathogenic antigen was not necessary for the production of specific antibodies, but this knowledge had not been applied in the production of antibacterial sera.

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[1] Arnold, H. D.: Boston Med. and Surg. Jour., 1909, clxi, 457. Medical Communication, Massachusetts Medical Society, 1909, xxi, 631. Tr. Am. Climat. Assn., Philadelphia, 1909, xxv, 128.

[1] Read at the meeting of the Tristate District Medical Society (Wisconsin, Illinois and Iowa), Rockford, Ill., September 5, 1919.

[2] Jour. Am. Med. Assn., June 24, 1911, No. 25, lvi, 1878-1881.

The above announcement was made after laboratory experiments, both *in vitro* and *in vivo*, had demonstrated high antibody content in the serum and its protective action in laboratory animals, as detailed by the investigator in his original paper. The next step was to determine its therapeutic value in man.

Extensive therapeutic testing is the final proof of the value of any curative serum. This truism applies with equal force to antitoxin and to antibacterial sera. In the case of the former, however, there is the additional certainty that one is dealing with a definitely standardized product, checked against known lethal doses of toxin, and this certainty is felt by the investigator both during the experimental work in the laboratory and in subsequent therapeutic testing. With a standardized product it is to be expected that definite results can be repeated. In the case of antibacterial sera, on the other hand, no accurate method of biological standardization has been devised. Protection against living organisms of varying virulence, rather than against known lethal doses of toxin, carries less conviction during the laboratory stage of investigation and makes therapeutic testing of even greater relative importance. The present report is made in this spirit. It is proper to state also that Kyes has not allowed control of production of the serum to pass from his hands pending such therapeutic testing, and that he still considers the serum in the experimental stage.

In 1918 Kyes[3] published the results of the use of his serum in the treatment of 115 cases of lobar pneumonia, controlled by a series of 538 cases, in which serum was not used, but otherwise treated under the same conditions, and during the same period of time. The mortality in the serum-treated cases was 20.8 per cent.; in the control cases, 45.3 per cent.

The next therapeutic test to which the serum was put in any large number of cases was at the Base Hospital, Camp Grant, Ill. An account of its use in the treatment of 322 cases between October 1, 1917, and September 20, 1918, has been published recently.[4] The death-rate was 7.7 per cent. While there were no control cases it was evident to the clinicians administering the serum that it had marked therapeutic value. In addition the series gave valuable data concerning dosage and methods of administration and other clinical phenomena, and afforded opportunity to improve the serum further on the basis of therapeutic use. With the present report the above constituted a complete record of the use of the serum in pneumococcus pneumonia at this Base Hospital. Mention may be made also, for sake of completeness, of five recoveries from pneumococcus meningitis during the epidemic following combined intraspinous and intravenous administration of the serum.[5]

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[3] Jour. Med. Res., July, 1918, No. 3, xxxviii, new series; No. 3, xxxiii, 495–501.

[4] McClellan, John H.: Jour. Am. Med. Assn., June 28, 1919, No. 26, lxxii, 1884–1885.

The present publication carries the record on from September 20, 1918, to May 1, 1919. It is properly divided into the epidemic period, during which it was possible to compare results in cases treated with and without the serum, and the period from the close of the epidemic until May 1, 1919, during which all cases received serum treatment.

There was very little question at Camp Grant as to the character of the infection with which we were dealing during the epidemic. The preponderating organism obtained by swab culture from the nose and throat, and by culture from the sputum, during the stage of so-called influenza, was determined after a very few days to be an organism of unusual virulence, conforming in all laboratory characteristics to the pneumococcus. The Pfeiffer bacillus was found in only a negligible number of cases. Pneumococci were obtained in pure culture, or preponderatingly, from the heart blood and organs of cases dead from pneumonia and from other forms of pneumococcus disease in a large proportion of the 198 autopsies done during the epidemic period.[6] It seemed to us that no doubt could arise as to the causative organism either in the cases of so-called influenza or of pneumonia, and that in the virulence of the organism was to be found sufficient explanation of the type of pneumonia resulting. It was presumed, therefore, that treatment with serum would demonstrate therapeutic efficiency.

Since it is proposed to compare the mortality-rates in two groups of cases, those treated with and those treated without serum, the conditions under which the serum was administered should be noted. There was no preparation for control comparison. The serum had been used routinely in the hospital and the propriety of withholding an agent of presumed therapeutic value from any cases for purposes of comparison was considered doubtful under the circumstances. But when it became apparent that there would shortly be more cases of pneumonia than serum could be furnished for, it was decided to restrict its use to cases in a single ward for ease of administration until more serum should become available. Its use was extended later to a second ward. Cases were admitted to these wards as fast as vacancies occurred, either directly from the receiving office or by transfer from so-called influenza wards by other individuals than those interested in or responsible for the treatment. There was therefore no possible selection of favorable cases. These wards were equipped in no essential way differently from the other pneumonia wards in the hospital. The serum was withheld from no cases in these wards. Finally, all deaths in the serum-treated group, except six that were determined at autopsy to have been due to streptococcus pneumonia, were charged against the serum, in spite of complications or accompanying diseases which might have contributed to or have actually caused death, and regardless of the length of the time between serum treatment and the time of death. Four cases admitted between September 26 and 30 died in November; two admitted September 26 died in December; one admitted September 30 died the following February.

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[5] Litchfield, L.: Jour. Am. Med. Assn., May 10, 1919, No. 19, lxxii, 1345–1348.

[6] Hirsch, Edwin F., and McKinney, Marion: Epidemic of Bronchopneumonia at Camp Grant, Ill., a preliminary report, Jour. Am. Med. Assn., November 21, 1918, lxxi, 1735–1736; and an Epidemic of Pneumococcus Bronchopneumonia, Jour. Infect. Dis., June, 1919, No. 6, xxiv, 494, 517.

Another circumstance especially unfavorable to the serum record should be explained. On October 1 it was decided to rearrange cases in three contiguous wards, transferring all acutely sick cases in Wards 12 and 13 to Ward 11, putting cases of apparently good prognosis into Ward 12, and convalescent cases into Ward 13. Ward 11 thereafter was to be the receiving ward for the group of three, from which patients were to progress as they improved. This was to protect patients moderately sick and conscious of their surroundings from the sight and sound of the dying. The same plan was put into operation in another group of wards a few days later, where the serum, which had now become available in larger quantity, was being used. It was proposed to extend the plan to the entire hospital, but the epidemic soon became so overwhelming that it was not carried further. Carried on in these two groups of wards without any thought of its effect on the serum record, for the serum was given to all cases admitted and transferred to these receiving wards irrespective of the stage of the disease, it is fair to state that the mortality was especially high among these transferred cases. However, these statements are not made as a basis for altering the death-rate of the serum-treated cases, and no figures have been compiled to show what might have been the death-rate if the serum had been administered to all cases on diagnosis. They are made to emphasize the fact that the circumstances were in no way made favorable for the serum-treated cases.

During the epidemic period 234 cases of pneumococcus pneumonia were treated with the serum. The number of deaths in this group was 39, a death-rate of 16.7 per cent. During the same period a total of 2064 diagnoses of pneumonia were made in the hospital. There was a total of 1088 deaths. The death-rate was approximately

52.7 per cent., but the total number of diagnoses and the total number of deaths include serum-treated cases and streptococcus cases, which must be subtracted for the purpose of this comparison.

Hirsch and McKinney,[7] in a paper previously referred to, detailed the bacteriology found at 198 autopsies during the epidemic. Streptococci were found in a small percentage. It was apparent that clinical differentiation between streptococcus and pneumococcus pneumonia had been largely impossible during the epidemic, partly because of the type of the epidemic diseases and partly because the necessary laboratory investigation for such a large number of individual cases was clearly out of the question. The total number of diagnoses of pneumococcus pneumonia recorded in the hospital, therefore, included a certain number of streptococcus cases. But since six deaths are cut out of the list of serum-treated cases because it was determined at autopsy that streptococci were the primary invading organisms, it becomes necessary to estimate a fair reduction in deaths among cases not treated with serum, few of these cases having come to autopsy. Using the same proportion as obtained in the serum-treated cases it is estimated that 140 deaths due to streptococcus pneumonia occurred among the cases not treated with serum. With the figures thus obtained the mortality in these cases, as detailed in the accompanying tabulation, is figured to have been 53.6 per cent.

The comparison I wish to submit, therefore, is a mortality of 16.7 per cent. in 234 serum-treated cases of pneumococcus pneumonia, with a mortality of 53.6 per cent. in 1684 similar cases treated in other respects by the same methods except that they received no serum. In other words the death-rate in the serum-treated cases was somewhat less than one-third that in the cases compared.

The serum was administered intravenously in all cases. Shortly before the epidemic the dose was increased from 2.5 c.c. once or twice daily to 5 to 10 c.c. twice daily, and this larger dosage was maintained during the epidemic. In a few cases as much as 30 c.c. was given daily. Commonly a total of 60 to 90 c.c. was given in cases that recovered.

As to the clinical phenomena following use of the serum little more can be said than is told in the reduction of mortality. No marked changes were regularly seen after serum administration that were not seen in individual cases not treated with serum. It was the feeling of the writer, therefore, that any attempt to tabulate symptomatic changes in the serum-treated cases for comparison with control cases would bring personal impressions into the record to such an extent that any conclusions arrived at would be open to question. Reduction of mortality, however, is a definite phenomenon. Therapeutic value of any treatment must finally be judged by this end-result.

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[7] An Epidemic of Pneumococcus-Broncho-Pneumonia, Jour. Infect. Dis., June 919, No. 6, xxiv, 594–617.

The *modus operandi* of the serum is, of course, of great interest but except for the fact that the serum is antibacterial any statement would be purely hypothetical. As to the so-called foreign protein reaction it should be stated that in only approximately 5 per cent. of cases was there any appreciable reaction following the serum used during the epidemic. This non-specific reaction cannot, therefore, be considered a factor in the reduction of mortality.

Following the epidemic, cases of pneumonia were atypical. Streptococcus pneumonia was much more common than during the epidemic. But pneumococcus pneumonia itself was also atypical, conforming in symptomatology neither to lobar pneumonia nor to the epidemic bronchopneumonic type, though the appearance of the lungs at autopsy was more clearly that of the latter. There was evidence that the organism was dropping in virulence (unpublished observation of E. F. Hirsch). Finally, in March and April, 1919, lobar pneumonia began to reappear. During the period, therefore, from the close of the epidemic to May, 1919, when this record closes, there was a continually changing picture of disease in the pneumonia wards. It was difficult to establish a clinical standard of differentiation of streptococcus from pneumococcus cases.

But while this period was in many ways an unsatisfactory one in which to attempt to evaluate the therapeutic effect of the serum, not only because of lack of controls but also because of the facts above set forth, it is believed that the final compilation of pneumococcus cases was accurate and that the low mortality is credited properly to the serum. There were 5 deaths among 118 serum-treated pneumococcus cases, a death-rate of 4.3 per cent.

The record of the use of Kyes's antipneumococcus serum at Camp Grant can now be summed up. 322 cases of pneumonia were treated between October 1, 1917, and September 20, 1918, with a death-rate of 7.7 per cent.; these were cases of typical lobar pneumonia. 234 cases of epidemic pneumococcus-broncho-pneumonia were treated between September 21 and November 4, 1918, with a death-rate of 16.7 per cent. which is to be compared with a death-rate of 53.6 per cent. in 1684 control cases. 118 cases were treated between November 5, 1918, and May 1, 1919, with a death-rate of 4.3 per cent.; these were atypical cases of pneumococcus pneumonia, which seemed to indicate a gradual change from the epidemic type of disease back to the type of lobar pneumonia, probably due to a falling virulence in the organism. In addition 11 cases of pneumococcus meningitis were treated, with 5 recoveries, 1 fatal case having occurred since the publication by Litchfield (loc. cit.)

EPIDEMIC STATISTICS, SEPTEMBER 20 TO NOVEMBER 5, 1918.

Total number of diagnoses	2064
Total deaths	1088
Death-rate	52.7%
Serum-treated cases	240
Streptococcus deaths subtracted	6
Pneumococcus cases	234
Pneumococcus deaths	39
Death-rate	16.7%
Non-serum cases	1824
Streptococcus deaths, subtracted (estimated)	140
Pneumococcus cases	1684
Pneumococcus deaths (see below)	903
Death-rate	53.6%
Total deaths	1088
Serum deaths	45
Non-serum deaths	1043
Streptococcus non-serum deaths (estimated)	140
Pneumococcus non-serum deaths (see above)	903

INCIDENCE, ASSOCIATED PATHOLOGY AND CLINICAL DIAGNOSIS OF CHRONIC PERICARDIAL ADHESIONS.

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The report of this investigation deals with a condition which, during the years recent and long passed, has received deliberate study from some of the most noted clinicians and pathologists, while apparently being seriously neglected by others equally noted. I refer to that pathological state characterized by an obliteration of the pericardial cavity, completely or in part, by adhesions. Associated with this there may or may not be extrapericardial adhesions. Because the adhesions associated with a pleural tuberculosis have a distinct clinical picture peculiar to this type of adhesions, we have sought, in so far as we were able, to exclude all cases possibly tuberculous from the series.

Since the study of this subject has been pursued only along lines of my own personal choice, no reference will be made to the not voluminous but rich and select literature. To the writer the two most perplexing features of this pathological state are its etiology and diagnosis. Many other intricate and interesting features surely do exist and will receive in their turn some discussion in which we may suggest that these associated features either explain the etiology or assist in rendering a diagnosis.

In the analysis here presented no definite theory as to the etiology will be advanced, as, even after a rather critical survey of the past histories of the cases representing this series, no specific etiology